marked symptoms than the lesions of tabes, etc , in the lumbar region. The clinical symptoms most frequently observed are, loss of the knee-jerks, pains, paræsthesia, ataxy, muscular weakness. A table of the seventeen cases investigated concludes this excellent paper, which well deserves being read in the original. STIEGLITZ.

Anatomical Contribution to the Study of Combined Systemic Diseases of the Cord.—Jakob (*Zeitschr. f. Nervenheilk*, Vol. 6, Part 1 and 2, 1894.

A report of the anatomical examination of a cord found accidentally amongst the specimens of the pathological laboratory in Erlangen. The clinical history of the case could not be obtained. The cord had been preserved for years in alcohol of 70%, after having been hardened in Müller's. The staining was done after Weigert-Pál, with neutral carmine, alum-carmine, and hæmatoxyein-eosin.

The examination showed: 1. Degeneration of the direct and crossed pyramidal tracts; the former up to the level of the upper lumbar region, the latter as far down as the conus. The intensity of the degeneration has been found to increase from above downwards. 2. Total degeneration of the cerebellar tracts in the whole length. 3. Incomplete degeneration of the columns of Goll. 4. Incomplete degeneration of the columns of Burdach. 5. The anterior and lateral roots are normal; the lateral and posterior root zones degenerated here and there. 6. Anterior horns normal, except a slight diminution of the fibres taking a transversal course. Clark's cells normal; anterior roots normal; posterior roots show some disease. The degeneration of the posterior columns does not reach so far down as the degeneration of the lateral columns All degenerated areas are symmetrically distributed on the cross section of the cord.

These findings lead the author to the following conclusions:

The symmetry of the diseased areas, their anatomical course and the absence of a primary focus in the whole extension of the cord, justify us in regarding combined systemic degenerations as a disease originating primarily in the nervous, and not in the accessory tissue of the central nervous system, *i. e.*, the disease starts in the nerve fibres proper and not in the vessels, neurolgia or meninges of the cord. The absence of a distinct myelitic focus militates against the theory of it being a focal or diffuse myelitis.

The symmetric distribution of the diseased areas is the expression of the elective regularity of the process, following the same path as the embryonic development of medullary sheaths to the fibres. Both ways the function of the fibres seems to have some importance; fibres of similar or allied function getting attacked by the disease and getting simultaneously medullated.

The histological nature of the process has been difficult to ascertain because of the fact that the cord was so long in alcohol. The signs of absolute inflammation found in the environments of the degenerated foci are secondary. The increased demand exercised on the resting fibres during the development of the disease might produce the dilatation of the vessels and the removal of the products of degeneration can be looked upon as a sort of mechanical or chemical stimulus. Noteworthy is the absence of disease in Clark's cells in spite of the total degeneration in the cerebellar tracts. FRAENKEL.

CLINICAL.

The Disturbances of Sensibility of the Globe of the Eye, and its Appendages in Locomotor Ataxia.—Berger (*La Méd. Mod.*, November 2, 1894). The disturbances of sensibility of the skin in locomotor ataxia are well known, but those of the visual sphere seem to have escaped the notice of the authors up to the present time. In the area of distribution of the trigeminal nerve, some importance, diagnostically, has been attached to anæsthesia of the pharynx, and peri-orbital anæsthesia of the skin, which, latter, is found in certain cases of tabic atrophy of the optic nerve. The following cases have been observed by the author: Observation I. Woman, 52 years old, had three delicate children and one miscarriage; no history of lues, but had periostitis of the cranium, cured by K. I. Present condition.—Symptoms of tabes well marked, diplopia, and paresis of the accommodation muscles. Tactile anæsthesia of the inferior papebral and bulbar conjunctiva. Observation II: Man, 46 years old, weak constitution. No history of nervous disorders or lues. Has a healthy daughter. Condition present in March, 1894—general paralysis, complicated with tabic symptoms. August 10, 1894—Mydriasis of left pupil; reaction to light and accommodation present in the right eye; in the left, the reaction to light is absent, to accommodation present (unilateral Argyll-Robertson's sign). Atrophy of the optic nerve; no Romberg's sign; knee-jerk absent on the left side; diminution of sensibility of the cornea in certain places; impaired localization of tactile sensibility over the skin of the nose, cheek, temples and infra-orbital region. Observation III: Strong man, 72 years old. Had gonorrhœa several times, no syphilis. For twenty years suffered from shooting pains. August 10, 1894—No knee-jerk on the right side; on the left, diminished; bilateral myosis; Argyll-Robertson's sign present; acuity of vision diminished in both eyes; optic atrophy. On the right side, anæsthesia of the inferior palpebral and bulbar conjunctiva; impaired localization over the skin of the upper lid, the forehead, and the temples. On the left side anæsthesia, of the temporal portion of the inferior palpebral conjunctiva; defective localization of the skin over the temporal region of the forehead and cheek. Observation IV. Weak man, 55 years old, with hereditary neurotic taint. In 1871 had indurated chancre, followed by an erythematous eruption; underwent an anti-luetic treatment. Was well until 1887, then it was observed that his character began to change, he becoming very impressionable. Since had headaches; and in 1891 sciatica, disagreeable sensations in and around the eyes, and atrophy of the optic nerve. Was accustomed to smoke fifteen to eighteen cigars daily. July 24, 1891—Romberg's and Westphal's sign present on both sides (two years previously the latter was only unilateral), tremor of the hands and tongue, convergent strabismus (due to paralysis of the sixth nerve on the right side and paresis of the same on the left); never had diplopia; bilateral myosis; Argyll-Robertson's sign; visual field diminished; anæsthesia of the palpebral conjunctiva, and diminution of the corneal sensibility (no reflex on touching the cornea); impaired localization of tactile sensations over the skin of the lids, temples, forehead, and cheeks. A peri-orbital gummata, 1 to 2 ctm. in width, is anæsthetic to the touch. Observation V: Weak man, 33 years old. Suffered from general nervousness and insomnia, following the application of cocaine for operation in the nose. In 1892 Professor Haab, in Zürich, detected atrophy of the optic nerve. Condition present August 4, 1894—Bilateral diminution of the visual field; atrophy of the optic nerve; anæsthesia of the palpebral and bulbar conjunctiva of both eyes; perceptions of heat and cold absent; impaired localization of sensations in the upper part of conjunctiva, and over the skin of the temples. Conclusions: In locomotor ataxia various troubles may develop in the eye and its appendages. Thus, in the cornea there are a diminution of sensibility, and disturbances of localization of tactile sensation. In the conjunctiva there is partial anæsthesia to the touch and temperature, also impaired localization. The skin of the lids may show disturbances of localization. The surrounding parts of the eye may also be impaired, there being present, peri-orbital anæsthesia, disturbances of localization over the skin of the forehead, temples, cheek, and nose. Summary: 1. There is no relation between the symptoms, above enumerated, and atrophy of the optic nerve of the tabic origin, there being cases in which the latter is present, with no disturbances of sensibility of the eye-ball and surrounding parts. On the other hand, these disturbances may be present in a tabetic subject without the co-existence of optic atrophy. 2. The presence of the disturbances of sensibility and localization, above enumerated, found in a case of atrophy of the optic nerve, point to a tabetic origin of the atrophy. MACALASTER.

Cerebellar Heredo-Ataxia.—Under the above head, Dr. Pierre Marie has published two papers in French medical journals; he first described and named the morbid syndrome, after putting together and interpreting several observations of his own and of Fraser, Sanger, Brown, Nonne, and Kleppel and Durante.

Paul Londe has recently recorded two cases of the same disease, collected few new observations, and published on the subject a monograph of 250 pages, "Heredo-Ataxic Cerebelleuse, Paris, 1895." The disease is a hereditary and family disease, one or more of the ascendants being neurotic and ataxic; more than one of a family also being affected in the same way. It is an ataxic disease, being attended with inco-ordination of movements and a staggering gait. It is also believed to be a cerebellar disease, the ataxia and inco-ordination being referred to a definite lesion of the lesser brain, viz., atrophy of the cerebellar cortex, or more particularly, of the cells of Purkinje. There have, however, been too few well-conducted autopsies to warrant very positive conclusions, though enforced by physiological considerations. The following observation of Londe and Brissaud brings out the salient points of this morbid syndrome: Patient, German, 24 years old; married; is of a neurotic family; died with some nervous diseases attended with trembling. Patient's brothers and sisters all "nervous; one is affected like her. For a year patient has had some difficulty in walking, especially in the dark. On presenting herself at the clinic it was noticed that she staggers.

Walks with head bent forward to see where to place her feet. A swinging pelvic gait (*elle marche du bassin*).

In the standing posture the oscillations of the trunk are constant; not increased by shutting eyes. With eyes open patient cannot stand on one foot. The head oscillates in rotation or flexion, especially when patient is under strong emotion. The speech is thick, jerky, a monotone. Exaggerated buccal contractions (as at angles of mouth) during articulation; no trembling of tongue; nystagmus when patient is looking intently; general inco-ordination, even in arms, which are agitated with choreiform movements when she is standing or walking; shoulder jerks; cannot with eyes open touch tip of nose with finger; some intentional trembling of hands; cannot do needle work or other fine work; muscular sense is intact; realizes position of limbs; has sense of weight of objects, etc, but motion of equilibrium is lost. When she walks it seems to her that she is "too light," that her feet cannot support the body. Instinctively clings to something to support her. Floor seems soft, and gives way under her.

Patellar and wrist reflexes normal; plantar reflex *nil* to tickling; contact is perceived. There is a double dorso-lumbar curvature of the spine.

Negative signs.—Absence of the Argyll-Robertson sign; absence of paresis of ocular muscles; absence of contractures; absence of vertigo and diplopia; absence of fulgurating pains; no difficulty of deglutition; no trouble with sphincters; no visual troubles; intellect only a little weakened; patient is easily fatigued.

She has been rheumatic and hysterical (*grandes crises* twice).

The vertebral deviation and the ataxia began with the pregnancy and accouchment in 1891; from that date were first noted the staggering gait, the oscillations of the head, the tremblings of the hands, etc.